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Invasive explorations in children younger than 3 years

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ABSTRACT

Purpose: In children with drug-resistant focal epilepsy who are candidates for surgery, invasive exploration is sometimes required. However, this is being controversially discussed for children younger than 3 years. The question of its necessity, feasibility and its risks is often raised, since it concerns primarily lesional epilepsy and a lesionectomy might be proposed right away. However, this attitude does not take into account the specificities of epilepsy at this age, including poor specificity of electroclinical semiology and the ongoing myelination challenging the interpretation of magnetic resonance imaging (MRI).

Methods: We retrospectively studied the records of children with drug-resistant epilepsy who were younger than 3 years of age at the time of their invasive exploration at our institution from 2000 to 2009. We reviewed the clinical, imaging and electrophysiological data, and included post-operative outcome for those who underwent surgery.

Key findings: 26 Children met the inclusion criteria. All had drug-resistant epilepsy that started at an average of 5.2 months (range 0–20 months) with multiple daily seizures in all and developmental delay in 16. The average age at the time of exploration was 21.8 months (range 5–35). In 20 children, subdural electrodes in combination with two or three depth electrodes were implanted, and in six children aged over 2 years a stereo-electro-encephalography (SEEG) was performed. SEEG was considered technically difficult to achieve before the age of 2 years. The tolerance of invasive exploration was good with a 3% morbidity consisting of one subdural hematoma during exploration by subdural electrodes, evacuated without any particular sequelae. In 25 patients, the exploration permitted to propose a focal resection. The surgical intervention was in the frontal lobe in 12 cases, the parietal lobe in six, the occipital lobe in two patients, and the temporal lobe in one child who underwent an additional resection. Four children had a resection of two or three lobes. Five underwent a second surgery, following a second invasive exploration. Histologically, the resected tissue revealed focal cortical dysplasia in 21 cases (including three patients with tuberous sclerosis), two post-ischemic lesions, one dysembryoplastic neuroepithelial tumor, and one gangliglioma associated with dysplasia. The mean postoperative follow-up period was 51 months (range 4–110). For the children operated on twice, follow-up was counted from the second surgery on. Seventeen children (68%) had an outcome of Engel class 1. In five (20%), seizure frequency was significantly improved (Engel class 3). In two of three patients without improvement in seizure frequency (Engel class 4), a new SEEG is planned and the third is presently a candidate for hemispherotomy.

Significance: Invasive exploration is feasible, well tolerated and carries a low morbidity in children under 3 years of age. At this age, it is indicated for drug-resistant lesional epilepsy associated with developmental delay. It permits delineating the lesion, which is not possible with MRI. The choice of the technique is in part age-dependent. The discussion of its indication arises in the same way as in the older child.

1. Introduction

Despite advances in imaging, invasive electroencephalogram (EEG) is still required in a number of children with pharmacoresistant partial epilepsy in order to delineate the epileptogenic region and/or to determine its relationship to functional cortex.[1,2] In infants, however, a common belief is that it is technically not feasible and does not provide more benefit than a resection following non-invasive exploration. Yet presurgical exploration in infants poses unique challenges. Analysis of the seizure semiology is difficult since motor signs, versive movements, autonomic manifestations and loss of contact

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are the only signs easily identifiable. Subtle attacks frequently go unnoticed by the parents or the examiner. As for imaging, due to incomplete myelination, it is difficult to interpret.[3] In most cases, the detected lesion remains ill-defined.

2. Methods

Of the 200 children under the age of 15 years who underwent invasive exploration between 2000 and 2009 at our institution, 26 were younger than 3 years at the time of exploration. No infant was explored within the first 3 months of life, since in our experience, surgery for epilepsy carries unacceptable morbidity before the age of 3 months. Twenty had a combination of subdural electrodes and depth electrodes (electrodes DIXI® or ADTECH®), while 6 were explored with stereo-electroencephalography (SEEG) (electrodes DIXI® or ALCIS®) as described by Talairach et al.[4] In the youngest age group, the feasibility of stereotactically implanted depth electrodes is still limited, since a minimum bone thickness is required in order to anchor the hollow pegs for the electrode insertion and fixation. This can be determined by computorized tomography, but in general, infants younger than 2 years cannot be explored by SEEG for this reason. During their invasive exploration, all children were seen by a child neurologist specialized in epilepsy. During the recording period (mean: 5 days, range 3–14 days) the children were examined by an EEG technician and a child epileptologist. When it was considered necessary, electrode stimulations were performed for motor cortex mapping 16 cases. Stimulation parameters were applied according to the protocol described by Jayakar et al.[5]

We retrospectively reviewed the records of all children, taking into account clinical, imaging and electrophysiological data. The latter were reviewed by an experienced electrophysiologist (DT), different from the one who had interpreted the recordings initially (CJ). For the 25 children who underwent surgery, we took into account the surgical and histopathological data. Seizure and developmental outcomes were based on the last follow-up examination. We used Engel's classification system of postoperative outcome, although it is not well adapted for pediatric patients.[6]

During the same period, among children younger than 3 years, 11 patients underwent a focal resection without invasive exploration, 5 the disconnection of a hypothalamic hamartoma and 57 an hemispherotomy.

3. Results

All patients had multiple daily seizures (from five to over 100 per day), despite several drug trials. An underlying metabolic or degenerative disease had been excluded in all cases. Clinical data are summarized in Table 1. The population includes 26 patients, 10 girls and 16 boys. The age at seizure onset was on average 5.2 months (from birth to 20 months, median 3 months, standard deviation 5.2 months). Fifteen children had only partial seizures and 11 partial seizures associated with infantile spasms. All patients had an identifiable lesion on magnetic resonance imaging (MRI); two of the three patients with tuberous sclerosis had multiple lesions (patients #24 and 25) while the third one had only one visible focal cortical dysplasia on MRI (patient #23). All children had focal interictal abnormalities on EEG, consistent with the anatomical site of the lesion. In all patients, a video-EEG had been performed either at our institution or the center where the child was followed, recording several seizures whose clinical semiology and discharges were consistent with an origin at the site of the lesion. The mean age at the time of invasive exploration was 21.8 months (range 5–35, median 22, SD 4.9). Only four children had a normal neurological examination. Five had focal neurological signs with normal development, six showed focal neurological signs and developmental delay. Ten children had developmental delay without focal neurological signs. In one patient (patient #17), the epilepsy started with status epilepticus that lasted for 4 months. During this period, it was difficult to evaluate his cognitive state which had been normal before the onset of epilepsy. The site of the seizures was in the right hemisphere in 10 and in the left hemisphere in 16 children. We present the imaging and neurophysiological data in Table 2. In all patient interictal

Table 1
Clinical data.

Patient	Sex	Age at onset (months)	Age at exploration (months)	Seizure type	Neurological examination	Side of epilepsy
1	F	4	17	FS	LHH	R
2	M	2	8	FS and IS	LHH	L
3	M	Birth	5	FS and IS	Normal	R
4	F	Birth	13	FS	LHH, motor asymmetry	R
5	M	2.5	20	FS and IS	LHH, motor asymmetry, developmental delay	R
6	M	10	20	FS	Motor asymmetry	L
7	F	5	22	FS	Normal	L
8	M	2	12	FS	Postural delay	L
9	M	10	24	FS	Speech delay	L
10	F	7	35	FS and IS	Speech delay	L
11	M	1	25	FS	Speech delay, postural delay	L
12	M	3	28	FS and IS	Postural delay, motor asymmetry	R
13	F	8	34	FS	Developmental delay	R
14	M	3	33	FS and IS	Developmental delay	L
15	M	2	30	FS	Developmental delay, motor asymmetry	R
16	M	1	8	FS	Postural delay, motor asymmetry, LHH	L
17	M	18	22	FS	Status epilepticus, unrelevant	R
18	M	20	35	FS and IS	Normal	R
19	M	7	26	FS	Developmental delay	R
20	F	1	13	FS	LHH, motor asymmetry	L
21	F	3	12	FS and IS	Postural delay, motor asymmetry	L
22	M	12	34	FS	Normal	L
23	F	7	35	FS and IS	Speech delay	L
24	F	3	14	FS and IS	Postural delay	L
25	F	1	33	FS and IS	Developmental delay	L
26	M	2	10	FS	Postural delay, LHH	L

FS: focal seizures; IS: infantile spasms; LHH: lateral homonym hemianopsia (suspected by clinical examination).

Table 2
Imaging and neurophysiological data.

Patient	MRI	Interictal scalp EEG	Ictal scalp EEG	Exploration type	Interictal intracranial EEG	Ictal intracranial EEG	Electrical stimulations	Remarks
1	R occipital ill delimited FCD	R occipital	R occipital	SD	Occipital pole	Occipital pole	Not performed	Mesial areas poorly explored
2	L posterior ill delimited FCD	L parietal	L parieto-occipital	SD	Inferior lateral parietal	Inferior lateral parietal; mesial areas poorly explored	Not performed	Mesial areas poorly explored
3	R frontal FCD	R frontal	R frontal	SD	Frontal	Frontal	Motor mapping	Subdural electrodes at the limit of the electrophysiologically defined dysplasia
4	R temporo-parieto-occipital pi lesion	R temporo-parieto-occipital	R temporo-parieto-occipital	SD	Temporo-parieto-occipital	Temporo-parieto-occipital	Motor area not found	Mesial areas poorly explored
5	Possible R occipital FCD	R occipital	R occipital	SD	Temporo-parieto-occipital	Occipital	Not performed	Mesial areas poorly explored
6	L Frontal dysplasia	L fronto-central	L fronto-central	SD	Opercular	Opercular	Motor area not found	Epileptogenic zone more widespread than the visible lesion
7	L Parietal FCD	L parietal	L parietal	SD	Parietal	Parietal	Motor mapping	
8	L frontal FCD	L fronto-central	L fronto-central	SD	Frontal	Frontal	Motor mapping	No orbito-frontal and no temporal exploration
9	L Temporal pi lesion	L temporal	L temporal	SD	Temporal	Temporal	Not performed	Epileptogenic zone more widespread than the visible lesion
10	Possible L frontal FCD	L fronto-temporal	L fronto-temporal	SD	Frontopolar and orbital	Frontopolar and orbital	Motor mapping	
11	Anterior frontal FCD	L fronto-centro-parietal	L fronto-centro-parietal	SD	Frontal mesial and lateral	Frontal mesial	Motor mapping, evocation of seizures	
12	R frontal FCD	R frontal	R frontal	SD	Anterior and lateral frontal	Anterior and lateral frontal	Motor mapping	
13[a]	Possible lesion R temporal	R temporal	R temporal	SEEG	Multifocal	Multifocal	Not performed	
14	L frontal ill delimited FCD	L frontal	L frontal	SEEG	Frontal; clear cut delimitation	Frontal	Not performed	
15	R Frontal FCD	R frontal	R centro-parietal	SD	Frontal	Frontal; incomplete exploration	Motor mapping	
16	L occipital FCD	L occipital	L occipital	SD	Occipital, parietal and temporal posterior	Occipital, parietal and temporal posterior	Not performed	Epileptogenic zone more widespread than the visible lesion
17	R frontal FCD	R frontal	R frontal	SD	Frontal	Frontal	Motor mapping	Explored during status epilepticus
18	R post-central FCD	R central	R central	SEEG	Fronto-parietal	Parietocentral	Motor mapping; evocation of seizures	Epileptogenic zone more widespread than the visible lesion
19	R frontal FCD	R frontal	R frontal	SEEG	Frontal	Frontal	Motor mapping	No posterior limit
20	L occipital FCD	L occipital	L occipital	SD	Occipito-parieto-temporal	Occipito-parieto-temporal	Not performed	Epileptogenic zone more widespread than the visible lesion
21	L frontal FCD	L frontal	Frontal	SD	Frontal and widespread spikes	Frontal	Motor mapping	Epileptogenic zone more limited than the interictal spikes
22	L parietal tumor	Bilateral centro-parietal	Bilateral centro-parietal	SEEG	Parieto-fronto-insular	Parieto insular	Motor mapping	Epileptogenic zone more widespread than the visible lesion
23	Probable FCD L parietal	L central	L central	SEEG	Parietal; clear cut delimitation	Parietal	Motor mapping	Epileptogenic zone more widespread than the visible lesion
24	L parietal mesial; other lesions (TS)	L temporo-parieto-occipital	L temporo-parieto-occipital	SD	Parietal; clear cut delimitation	Parietal	Motor area not found	
25	Multiple FCD including L frontal; other lesions (TS)	L frontal	L frontal	SD	Frontal (multifocal) and temporal	Frontal (multifocal) and temporal	Not performed	Multifocal; prognostic restrictions for surgery
26	L temporo-occipital	L temporo-occipital	L temporo-occipital	SD	Parieto-occipital sparing temporal	Parieto-occipital	Not performed	No temporal (abnormal on MRI)

R: right; L: left; FCD: focal cortical dysplasia; SD: subdural exploration; pi: post-ischemic; TS: tuberous sclerosis.
[a] Recused for surgery.

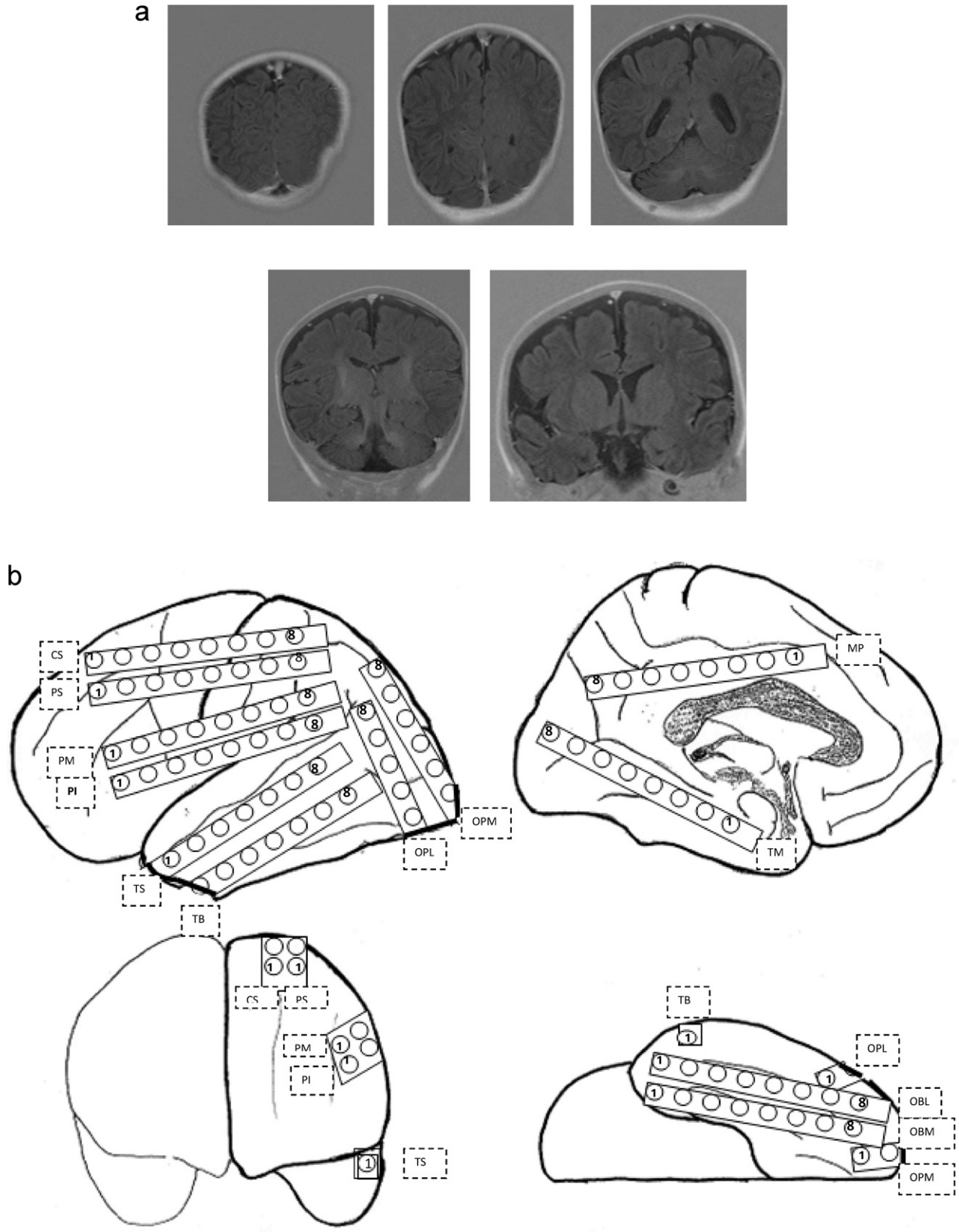

Fig. 1. Subdural exploration at patient #20, 13 months old at time of exploration. This figure shows that electrophysiological delimitation of focal cortical dysplasias is more precise than imaging. (A) MRI at 3 months of age showing a probable focal cortical dysplasia left parieto-occipital. The extension is unclear, in particular in temporal lobe. (B) Schema of the electrode implantation. (C) Seizure type 1: the child opened the eyes and had an eye deviation to the right after the onset on TM6-8 (mesial occipital) (noted as SO) 1D: seizure type 2: behavioral change after the onset on BM 1–4 (temporopolar) (noted as SO).

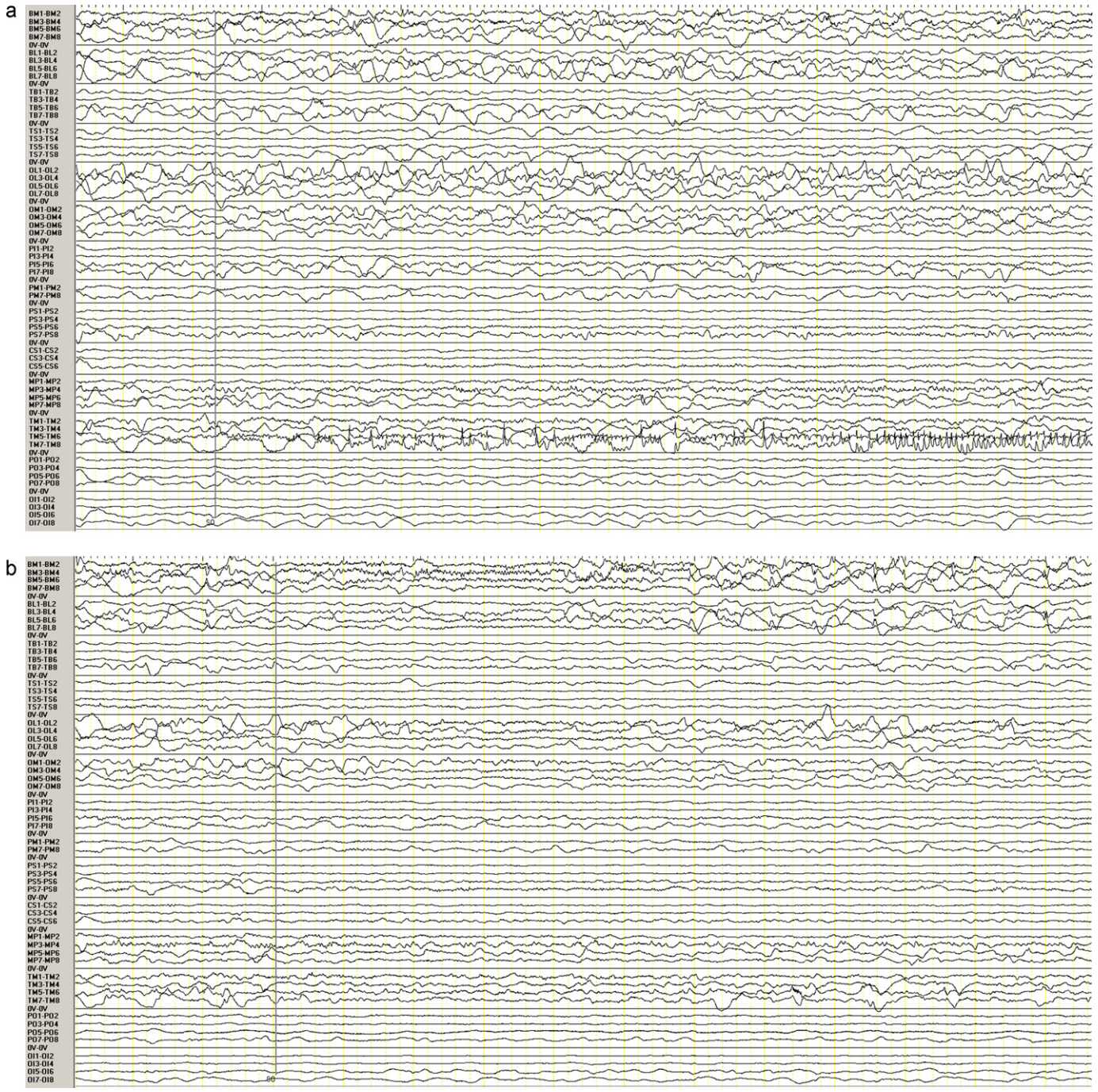

Fig. 1. (*Continued*).

continuous spike-waves were seen. In most cases they had a complex organization with several focus in the same brain area. The ictal pattern was made of a rhythmic activity with spatial and temporal evolution correlated to clinical features. Different seizures pattern were recorded within the epileptogenic zone. An illustrative case is presented in Fig. 1.

In all cases but one, electrode implantation and long-term invasive video-EEG monitoring were well tolerated. In one child (patient #7), a subacute subdural hematoma following craniotomy with subdural grid implantation was surgically removed without compromising the pursuit of the recording and without any neurological sequelae. In only one patient (patient #13), invasive recording revealed multifocal seizure onset precluding surgical treatment. In the other 25 children, we could determine a seizure focus, which was followed by a functional mapping through cortical stimulations which were judged to be necessary in 16 cases. In 3 patients (19%) no motor response could be evoked. In all 25 children, focal surgery was performed (Table 3). In 24 patients the ictal onset zone was considered to be removed by surgery. In patient #25 suffering of tuberous sclerosis rare seizures originated out of the main ictal onset zone. We decided however to offer surgery, hoping to cure the more frequent seizures.

Mean age at surgery was 23 months (range 5–65, median 22, SD 4.9). In 21 it consisted of cortical resection, in one patient disconnection was performed, in two a combination of resection with disconnection, and in one child a cortical resection associated with multiple subpial transsection of functional cortex. Surgery concerned the frontal lobe in 12 cases, the parietal lobe in six, the

Table 3
Surgical and follow-up data.

Patient	Age at surgery (months)	Surgery type	Post operative complication	Pathology	Follow-up (months)	Engel's class	Schooling
1	17	O	No	Dysplasia type IIb	66	1	Primary school
2	8	P	Expected quadradanopsia	Dysplasia type IIb	4	1	Secondary school
3	5	F	Paresis of left arm	Dysplasia type IIb	110	1	Primary school
4	13	TPO (resection disconnection)	No	Post-ischemic lesions	102	1	SES
5	20	O	Expected hemianopsia	Dysplasia type IIb	13	1	SES
6	20	F	No	Dysplasia type IIb	100	1c	Primary school
7	22	P	No	Dysplasia	88	3	Primary school
8	12	F	No	Dysplasia type IIb + HS	79	1	Primary school
9	24	T	No	Post-ischemic lesions	77	1	Primary school
10	35	F (disconnection)	No	Dysplasia	83	1	Primary school
11	25	F	No	Dysplasia type II	67	1	SES
12	28	F	No	Dysplasia type IIb	65	1	SES
14	65	F	No	Ganglioglioma associated with dysplasia (type IIIb)	48	1	SES
15	30	F	No	Dysplasia type IIb	58	3	SES
16	8	OP and temporal posterior	No	Dysplasia type IIb	65	1	Too young
17	22	F	Subdural collection treated with shunt	Dysplasia type IIb	44	3	Too young
18	39	P (resection and subpial transsections)	No	Dysplasia type IIb	28	1	Primary school
19	33	F (resection disconnection)	Paresis of left arm	Dysplasia type IIb	28	4	Too young
20	13	TPO	No	Dysplasia type IIb	29	1	Too young
21	12	F	Subdural collection treated with shunt	Dysplasia type I	32	4	Too young
22	46	P	No	DNT	12	1	Too young
23	40	P	Right hemiparesis	Dysplasia type IIb	9	1	Too young
24	14	P	No	Dysplasia type IIb	20	3	Too young
25	33	F	No	Dysplasia type IIb	13	3	Too young
26	10	PO	No	Dysplasia type IIb	24	4a	Too young

T: temporal; F: frontal; P: parietal; O: occipital; HS: hippocampal sclerosis; DNT: dysembryoplastic neuroepithelial tumor. In bold, patients operated on twice. For them, the follow-up period was calculated from the second surgery on. Sex: M, male; F, female; SES: special education system.

temporal lobe in one, and the occipital lobe in two. In four children, surgery was multilobar (parieto-occipital in one and temporo-parieto-occipital in three). Complications of curative surgery are listed in Table 3. Three patients had motor deficits probably related to ischemic complications and not to the resection of motor cortex; 2 had subdural collections which required a shunt. Two children had expected visual field defects which cannot be considered as complications.

Histopathological examination revealed cortical dysplasia (FCD) in 21 cases. According to Blümcke et al.'s classification, it was, type 1 in one case, type 2a in one case and type 2b in 17 cases but not further specified in two cases.[7] Of the 16 patients with FCD type 2b, 3 had a known tuberous sclerosis. In one patient (patient #8) who was operated twice, the first time in the frontal lobe, the second time in the fronto-temporal lobes, frontal FCD type 2b had been only partially resected during the first surgery, and was associated with hippocampal sclerosis probably related to several severe status epilepticus that the patient had suffered from in the past. A dysembryoplastic neuroepithelial tumor (DNT) was diagnosed in one patient, a ganglioglioma associated with dysplasia was not further classified in another patient, whereas two patients had post-ischemic lesions. The mean postoperative follow-up period was 51 months (range 4–110, median 51, SD 32.8). For children operated on twice, the follow-up period was calculated from the second surgery on. Seventeen children (68%) became seizure-free (Engel 1). Of these, 12 had no seizures from the surgery on. In the other five patients with persisting seizures, a second invasive exploration was performed, in all by means of SEEG, since in our view, a new craniotomy and placement of subdural grid electrodes after earlier brain surgery is associated with a higher complication rate compared to the SEEG technique. In these five patients, the first surgery had resulted in seizure reduction and improvement of psychomotor development. In four cases, the remaining epileptogenic zone was located to the mesial cortex of the lobe(s) poorly investigated during the first invasive exploration. In one case (patient #8), the frontal exploration had been too limited and the temporal lobe had not been investigated. The second surgical resection resulted in complete disappearance of seizures. The mean period between first and second surgery was 73.6 months (median 74, range 40–124, SD 33). The reason for this relatively long delay wished by the parents is related to the significant improvement in psychomotor development which all five children experienced following their first surgery. In two children, the second surgery consisted in limited additional cortical resection within the same lobe. In the three other children, bi- or trilobar resection/disconnection followed a former focal single lobe surgery. Those five patients were all explored before 2003; their evolution led us to explore more widely the mesial structures and this need of reexploration did not occur again since this time. In five patients (20%), epilepsy was significantly improved (Engel 3) with persisting seizures of short duration and very little social impact. Among them, one child could attend a regular school system. One of them had a second invasive exploration with SEEG, which disclosed a seizure onset within the centro-parietal region. Considering the functional risk, a second surgery was therefore not planned. Three patients had no significant postoperative improvement in seizure frequency (Engel's class 4, 12%). In two of them, a new exploration with SEEG was planned. In the third patient, multifocal seizures within the left hemisphere have evolved over time and hemispherotomy was planned. Of the 15 school age children, 9 (60%) were attending a regular school and six were in a specialized education system.

4. Discussion

Lesional epilepsy in the newborn or infant has specific characteristics that need to be considered in order to determine the therapeutic approach. The severity of epilepsy must be judged not only on frequency and type of seizures, but also the occurrence of status epilepticus and, notably, the impact on psychomotor development. The concept of drug resistance commonly used for adults cannot be applied in the same way. Side effects of medical treatment must also be taken into account. In case of rapid depletion of medical treatment options, early surgery appears to be the only chance for the patient. The indication is the subject of a consensus,[1] and surgery should be considered without any delay. Worldwide, few teams practice epilepsy surgery in young infants and only few series have been reported to date. Convincing results have been confirmed more recently by several authors in children having like our patients MRI visible lesions.[8–10] Although the indication for surgery is no longer discussed, the role of invasive explorations to achieve satisfactory results following cortical resection is not codified. Although subdural explorations were shown in the past to be a safe technique in this age group,[11] they are not used by the three teams mentioned above. However, as for patients in any other age group, the rationale from the epileptologist's point of view, in order to propose a cortical resection, requires a clear anatomo-electroclinical correlation. Such correlation is particularly challenging in young children, owing to the difficult interpretation of clinical seizure semiology and the imaging. In addition, cortical resections are extratemporal in the majority of infants. All these arguments led us to propose invasive exploration in children younger than 3 years even more often than in older patients.

At our institution, we have performed presurgical invasive explorations in infants since 1995. In the present study, however, in order to have better homogeneity with respect to the process of decision-making and available neuroimaging, we restricted the study to patients who were explored within the last 10 years. In this series, the electrode implantation did not present any particular difficulties for the pediatric neurosurgeons. The tolerance of the invasive explorations was good with a 3% morbidity consisting of a subacute subdural hematoma occurring during the exploration by subdural grid electrodes, which was evacuated without any particular consequences for the patient or further recording. None of the children presented any surgery-related infection despite their young age. This may be due in part to the development of a surgical technique of wound closure following subdural grid placement, which consists in an individual subcutaneous tunnelization of each electrode to a certain distance from the craniotomy site, in order to reduce cerebrospinal fluid leakage during the recording period. We cannot compare our results with those of children we operated on without invasive explorations. As we are a tertiary center, over the same period only 11 children aged less than 3 years underwent a focal resection including 4 temporal lobe epilepsies, one Sturge Weber syndrome and 6 extratemporal epilepsies linked to different types of lesions. In our view surgical outcomes are not to be compared.

The EEG analysis does not pose particular problems for the electrophysiologist who is familiar with the reading of invasive recordings. Invasive explorations allow achieving functional cortical mapping, which is indispensable in selected cases. However, as previously noted in this age the motor area cannot be found in some cases (19% of patients in whom electrical stimulations were performed).[5] Motor mapping can also be performed intraoperatively, but in our experience the evoked responses can also be missing. The main objective of invasive exploration is to provide a precise electrophysiological delimitation of the epileptogenic focus, which is not possible based on MRI alone. The electrophysiological boundaries were quite precise but the complexity of the data makes unlikely to understand them with electrocorticography. It is not possible in our series to compare the results of invasive recording and MRI because the extension of the lesion on MRI is often ill defined. However the epileptogenic zone and the extension of the surgical resection go in general beyond the clear-cut part of the MRI abnormality. This is so obvious for the teams who had not recourse to invasive exploration that they frequently performed lobar or multilobar resections. This was the case in the series of Gowda et al.[9] where all patients underwent complete lobar or bilobar resection. Similarly, in another recently published series, only 10% had focal resection of less than an entire lobe.[10] Even when functional imaging techniques were included in the presurgical work-up, such as PET scan by Chugani et al.,[12] the extent of resection was no more restricted. This latter group published a surgical series, resulting in at least bilobar resections in 13 of 15 infants.[12] In our own series, including the cases with second surgery, only seven multilobar resections were performed. In our view, it is indeed preferable from a cognitive point of view to carry out limited resection whenever possible although no controlled data validates this view. In contrast two patients with tuberous sclerosis had multiple lesions which were not resected and at least in one patient (#26) had an abnormal temporal pole in MRI which was electrophysiologically normal.

Comparison of our results with recent series is difficult since they include patients with hemispheric surgery. Hemispheric surgery raises other questions, has a better seizure outcome and should not in our view be compared to focal resections. A huge advantage of the exploration consists of better understanding of the epilepsy and better analysis of failures. Indeed, this allowed us to offer, based on strong arguments, a new exploration and second surgery permitted complete seizure relief in all cases. This allowed also to better explore following patients of the series especially mesial cortex in parieto-occipital epilepsies.

At our institution, we use both subdural and intracerebral electrode explorations. The choice between the two techniques has been gradually refined with increasing experience and after 2 years of age the decision for the appropriate form of exploration is determined on a case by case basis. SEEG remains indispensable for the exploration of certain regions, such as the mesial cortex of the temporal, inferior frontal and parietal lobes, as well as the insular cortex. In the setting of functional mapping, extensive cortical coverage by subdural grid electrodes provides definitely more information than depth electrodes, particularly when functional anatomy can be expected to be displaced, such as in large cortical dysplasias of motor or language cortices.

In most infants explored, the very high seizure frequency permitted more than sufficient documentation. However, a prolongation of the recording is required from time to time, and for technical reasons, the SEEG technique is clearly superior to subdural grids with regard to the risk of infection.

In every case studied, the analysis and delineation of the epileptogenic zone should be developed in the three-dimensional space. In all children who are explored with subdural grids, we therefore require additional depth electrodes which are introduced under neuronavigation guidance or, more recently, with the aid of robot-guided frameless stereotaxy.

SEEG, which is generally less invasive and does not require surgical resection immediately after the recording, can be used for patients whose indication for surgery is less evident, as in the case of our patient #13. In these cases, like in patients with deeply located epileptogenic lesions (for instance insular or frontal mesial), it can be considered to have the patient wait several months in order to reach the age of possible SEEG. Drug-resistant lesional epilepsy associated with developmental delay

predominates in children younger than 3 years. The clear advantage of invasive recording is to obtain a complete delineation of the epileptogenic lesion, which cannot be achieved by MRI alone. From our experience, it is feasible, well tolerated and associated with a low morbidity in this age group.

Conflict of interest statement

None of the authors has any conflicts of interest to disclose.

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